CASE REPORT

# A Rare Diagnosis of a Common Problem: Acute Severe Low Back Pain with Paraplegia

Narayanan Sarat[1], Dedeepiya Devaprasad[2], Senthilkumar Rajagopal[3], Ramesh Venkataraman[4]

*Received on:* 07 July 2022; *Accepted on:* 13 July 2022; *Published on:* 31 August 2022

*Indian Journal of Critical Care Case Report* (2022): 10.5005/jp-journals-11006-0007

## Case Description

A 65-year-old gentleman with a past medical history of diabetes mellitus (DM), hypertension (HTN), coronary artery disease (CAD) with old inferior wall myocardial Infarction, normal left ventricular function, and chronic low back pain presented to our hospital with acute exacerbation of low back pain and acute onset of weakness of bilateral lower extremities (LE). Weakness started in left LE first and then progressed rapidly to bilateral complete weakness of both LE within 24 hours. The patient presented to our hospital after being intubated at an outside facility. On presentation to our hospital, he was conscious and obeying commands. He had an oxygen saturation of 100% on positive pressure ventilation. He had a pulse rate of 92/min with a BP of 160/100 mm Hg. Both his lower limbs were cold to touch and capillary refill time was >3s. His lower limb peripheral pulses including dorsalis pedis, posterior tibial, and popliteal arteries were not palpable on both sides. Neurological examination showed the power of 0/5 on both lower limbs with loss of muscle tone, absent deep tendon reflexes, and loss of sensation below the inguinal ligament. The rest of the examination was normal.

## Question 1

### What Information in History would You Like to Elicit to Identify the Possible Cause for this Presentation?

*Answer*

First, when eliciting a history of motor weakness manifesting as an inability to move the legs, it is important to also elicit a history of loss or alteration of sensations in the limbs and any history that suggests bladder and bowel involvement. Isolated motor weakness is usually a pointer to muscle or neuromuscular junction as the structures are likely to be involved. However, rarely isolated motor neuropathy can also present as pure motor weakness. Along with motor weakness, when there is also sensory and autonomic (bladder and bowel) involvement, the lesion is likely to be present in the peripheral nerve (or) spinal cord.

Second, in assessing motor weakness it is important to understand whether the weakness is symmetrical or asymmetrical. Symmetry helps us to understand where the weakness is likely from. Symmetrical lesions are more likely to be due to muscle or neuromuscular disorders. Asymmetrical motor weakness is likely to be a result of structural lesions that cause anatomical damage to related neural structure (e.g.) compressive lesions such as neurofibroma, disk prolapse, spondylodiscitis, etc. All these begin asymmetrically but may progress over a period of time to produce symmetric weakness.

[1–4]Department of Critical Care Medicine, Apollo Hospitals, Chennai, Tamil Nadu, India

**Corresponding Author:** Ramesh Venkataraman, Department of Critical Care Medicine, Apollo Hospitals, Chennai, Tamil Nadu, India, Phone: +91 4428296517, e-mail: ccmramesh@gmail.com

**How to cite this article:** Sarat N, Devaprasad D, Rajagopal S, *et al.* A Rare Diagnosis of a Common Problem: Acute Severe Low Back Pain with Paraplegia. Indian J Crit Care Case Rep 2022;1(1):16–19.

**Source of support:** Nil

**Conflict of interest**: None

Another useful information would be, whether there is preferential involvement of proximal muscle groups, distal muscle groups, or whether both groups are equally involved. Usually, proximal weakness is likely to be due to a muscle pathology while distal weakness is likely to be due to neuropathy, and involvement of both proximal and distal muscle groups implies radiculopathy.

The rapidity of onset of weakness would help in narrowing our diagnosis. Whether onset is acute—acute static or acute progressive, with progressive involvement of muscle groups should be clarified. The time of onset and progression of weakness would help in narrowing our diagnosis by establishing an etiology (i.e., what is causing the lesion?). While acute static weakness usually stems from spinal cord injury, acute progressive weakness implies a demyelinating disorder (e.g., Guillian-Barre syndrome), and a slower onset with gradual progression is seen in myasthenia gravis. Fluctuation of muscle weakness with easy fatigability points to neuromuscular junction lesions.

History of trauma and pain over the back points to a vertebral fracture causing spinal cord compression. History of fever with pain may point to an infective etiology. Epidural abscess due to *Staphylococcus* may present with an acute history of fever with chills, while epidural abscess due to tuberculosis presents as a low-grade fever of at least 2–3 weeks duration. History of recent fever or vaccination in the preceding few weeks raises the possibility of postinfectious demyelination (transverse myelitis) or acute inflammatory demyelinating polyneuropathy. History of recurrent embolic phenomena such as stroke, and mesenteric ischemia in the past should alert to the possibility of embolic spinal artery occlusion. Acute spinal artery occlusion usually involves the anterior spinal artery, rarely the posterior spinal artery but almost never both simultaneously. If this patient had undergone abdominal aorta aneurysm repair and developed complete motor paralysis and sensory loss with bladder and bowel involvement in the postoperative period, then this could be attributed to

bilateral ischemic plexopathy of the lumbosacral region. History of abdominal pain with back pain raises the possibility of either embolic showers causing the simultaneous occlusion of the mesentery (abdominal pain) and spinal arteries (motor weakness) or the compression by an expanding aortic aneurysm over the vertebra and spinal cord.

When dense motor involvement involves multiple levels, it can be due to a lesion in the spinal cord (e.g., transverse myelitis, vertebral cord compression), epidural space (epidural abscess, epidural hemorrhage), or arachnoid space (arachnoiditis). In these situations, it will be good to assess the individual myotomes as described briefly below to determine the level of motor involvement.

- L1 and L2 - Hip Flexion - Ask the patient to bring his heel to touch his gluteal region
- L3 - Knee extension - Ask the patient to imagine kicking a football
- L4 - Dorsiflexion of the ankle - Ask the patient to move the ankle upwards
- L5 - Great toe extension - Ask the patient to move his toe upward
- S1 - Plantar-flexion of the ankle - Ask the patient to move the ankle downwards.

## Question 2

### What Initial Examination Findings would be the Key in Narrowing the Differential Diagnosis?

*Answer*

The most important clinical task is to assess whether the limb is cold. A quick examination of the pulses of dorsalis pedis, posterior tibial, popliteal, and femoral arteries pulse will exclude arterial occlusion as a cause. Point of care ultrasonography with arterial doppler would be useful for the same. This is important as arterial occlusion as a cause of pseudoparalysis should be recognized immediately as delayed recognition leads to inevitable delay in revascularization and potentially irreversible adverse outcomes.

Simple bedside tests have an important role in this situation to help arrive at the diagnosis. The loss of pain (tested by pins) and loss of temperature (tested by cold and hot water-filled test-tubes) with preservation of vibration (tested by tuning fork) and preservation of joint position sense (tested by passively moving joints with the patient's eye closed) helps to identify an anterior spinal artery occlusion. The dorsal column sensations (vibration and joint position sense) are typically spared in this situation. However, all this is possible to test, only if the patient has a normal mental status and is cooperative. In addition, examination of deep tendon reflexes and plantar reflexes will enable differentiation of lesions at or above the spinal cord from those below.

## Question 3

### Can You Summarize the Key Points from the Initial History and Clinical Exam?

*Answer*

A middle-aged gentleman with a history of DM, HTN, and CAD presented with acute exacerbation of his low backache and acute onset of rapidly progressing weakness of both lower limbs. On examination, he was found to have absent peripheral pulses on both lower limbs with flaccid paraplegia and absent sensations.

## Question 4

### What are the Probable Diagnoses Based on the Presented History and Exam Findings?

*Answer*

The absence of the distal pulses along with motor weakness suggests likely embolic occlusion of the aorta iliac bifurcation as the most possible cause. Dissecting an aneurysm of the abdominal aorta extending to the iliac bifurcation and beyond can result in the false lumen occluding the true lumen leading to pulse loss and motor weakness.

## Question 5

### What are the Initial Investigations that would be Recommended Based on Our Initial Clinical Suspicion?

*Answer*

Initial investigations specific to the presentation should be focused on ruling out a vascular etiology and an acute spinal pathology. CT angiography of the abdomen to rule out major aortic pathology and MRI thoracolumbar spine to rule out spinal cord pathology would be the initial investigations of choice for this purpose.

In this patient, these two investigations were done on admission. His CT angiogram showed total occlusion of the distal aorta from the level of inferior mesenteric artery origin, aortic bifurcation, and bilateral common iliac arteries with thrombus. Also, there was a focal eccentric, mural thrombus in the aortic arch just distal to the left subclavian origin.

Magnetic resonance imaging (MRI) thoracolumbar spine showed diffuse posterior disk bulge seen indenting the thecal sac and abutting bilateral exiting nerve root. Bilateral facet joint arthropathy causes mild narrowing of bilateral neural foramina.

## Question 6

### How do You Explain the Symptoms in the Light of the Provided Diagnosis?

*Answer*

The pulselessness is explained by the aorto-iliac occlusion which leads to no arterial flow to the limbs. The back pain is explained by the lack of blood flow through the lateral sacral arteries and inferior and superior gluteal arteries which are all branches of the iliac arteries and supply the muscles in the gluteal region and back. The decreased tone of muscle, absent deep tendon reflexes (DTR), and absent sensations can be most likely explained by ischemia of the cauda equina, sacral nerve roots, and ganglia (which is usually difficult to image). This can also result from direct spinal cord ischemia but that can be usually detected on MRI imaging (absent in this patient making that possibility less likely).

## Question 7

### What are the Possible Interventions for this Acute Medical Emergency?

*Answer*

After initial stabilization, the first therapeutic option should include immediate therapeutic anticoagulation. Anticoagulation with unfractionated heparin infusion using a weight-based protocol

prevents propagation of thrombus and decreases the chances of new thrombus in the distal low-flow vessels.

The therapy of choice will depend on the etiology, location of the thrombus, extent of the thrombus, duration of symptoms, the imminent risk to the limb, and patient suitability and stability for any intervention. If the limb has an immediate threat, surgical thrombectomy would be preferable. In patients not amenable to surgery and in patients who have a marginal threat to the limb, intra-arterial thrombolysis may be an acceptable option.[1] Intra-arterial thrombolysis is done through a catheter placed directly into the vessel proximal to the thrombus and a thrombolytic agent (e.g., tissue plasminogen activator) is infused to break the thrombus. Patient conditions that contraindicate thrombolysis should be ruled out prior to initiation. In suitable cases, transcatheter embolectomy may be attempted. This is especially the case for intraprocedural embolizations. Endovascular treatment of occlusive disease has also been utilized in patients who are at high risk for surgery. Endovascular treatments have the advantage of faster recovery time, and less pain and morbidity. Hybrid procedures combining open and endovascular approaches are increasingly being adopted.[2] If the limb is nonviable—amputation of the limb at a suitable time should be considered.

## Question 8

### What are the Problems that should be Anticipated and Monitored for Post-therapy?

*Answer*

Monitoring of the patient should predominantly involve monitoring clinical features, predominantly signs of reperfusion of LE including gradually improving pain, warmth, color, and palpability of peripheral pulses. Simultaneously any evidence of bleeding should be observed for in patients receiving anticoagulants or thrombolytics. Signs of ischemia-reperfusion syndrome should be looked for. Commonly these include, increasing pain and swelling of the extremity with gradually increasing weakness suggestive of compressive neuropathy from compartment syndrome.

## Question 9

### What is Reperfusion Injury? What are the Mechanisms and How does it Clinically Manifest?

*Answer*

Reperfusion injury is secondary tissue injury caused when blood flow and oxygenation are restored to an organ that had a period of ischemia.

Ischemic injury to tissue leads to anaerobic metabolism leading to decreased ATP production and lactate-induced metabolic acidosis in the cells. Low levels of ATP cause failure of ATP-dependent Na-K and Ca pumps which leads to cell swelling secondary to the retention of sodium and impaired enzymatic activity in the cells.

In addition, when reperfusion occurs, oxidative stress occurs in the cells due to various mechanisms:

- Xanthine oxidase: In reperfusion, hypoxanthine is converted to xanthine and uric acid by xanthine oxidase along with the release of reactive oxygen species like superoxide and hydrogen peroxide.
- Nicotinamide adenine nucleotide phosphate (NADPH) oxidase: There is increased activity of NADPH oxidase and similar enzymes due to the release of chemical mediators like TNF-α, interleukins, phospholipase A2, etc. after reperfusion. These enzymes produce peroxide and superoxide ions.
- NO species: Nitric oxide combines with superoxide anion to form peroxynitrite radical which can promote lipid peroxidation and cellular membrane damage.

Oxidative stress then causes endothelial dysfunction, activation of inflammatory cascade including activation of cytokines, immune, and complement activation, and cell damage eventually leading to cell death. Cell damage and cell death in reperfusion injury occur by various processes including apoptosis, necrosis, necroptosis, and autophagy.[3]

The clinical manifestations of reperfusion injury can be diverse and varied from minimal local tissue injury to multiorgan dysfunction syndrome. Local injury depends on the organ affected. In limb ischemia, reperfusion injury can lead to muscle edema, necrosis, and compartment syndrome. Systemic complications include metabolic abnormalities like hyperkalemia, hypocalcemia, acidosis, rhabdomyolysis, acute kidney injury, systemic inflammatory response syndrome, acute respiratory distress syndrome, and multiorgan dysfunction syndrome.[4,5]

## Question 10

### What was the Rest of the Hospital Course for this Patient?

*Answer*

The patient was initially started on intravenous heparin infusion targeting a therapeutic partial thromboplastin time for the aortic thrombus. He underwent catheter-directed thrombolysis on day 2 of ICU admission. Alteplase at 1 mg/hour was given for 24 hours for the thrombolysis. Follow-up check angiogram postprocedure showed partial resolution of the thrombus and there was the restoration of peripheral pulses in the lower limbs. However, the weakness of the bilateral LE did not improve. He also underwent aortobiiliac and right renal artery stenting on day 5. All the procedures were completed uneventfully.

He developed acute kidney injury requiring regular dialysis from day 3 of hospital admission–probably secondary to rhabdomyolysis, reperfusion injury, and contrast-induced nephropathy.

Later, the patient developed recurrent sepsis with multisystem organ dysfunction syndrome secondary to an infected ischemic ulcer on his lower back which progressed to necrotizing fasciitis involving the lower back, large portions of both gluteal regions, and perineum extending into the scrotum. He had to be intubated and mechanically ventilated on day 9 for respiratory distress. Initial cultures from the ulcer biopsy grew *Morganella morganii* and *E. coli* on day 12 of their ICU stay for which he was treated with appropriate antibiotics (Meropenem and Tigecycline) and debridement. The tissue cultures grew *Providencia rettgeri* and *Morganella morganii*.

He also developed multiple secondary infections including ventilator-associated pneumonia (VAP) secondary to carbapenem-resistant *Acinetobacter baumannii* and catheter-associated urinary tract infection (CAUTI) secondary to *Candida glabrata* for which he received appropriate antimicrobials.

He was being treated with supportive care during the ICU stay with ventilator support, vasopressors, regular dialysis, and appropriate antibiotics.

Later, he was transferred to another hospital for further management on day 25 of his ICU admission. At the time of

discharge, he was on mechanical ventilation, requiring vasopressor support; had restored circulation to his lower limbs and power of 2/5 on his lower limbs.

## References


1. Results of a prospective randomized trial evaluating surgery versus thrombolysis for ischemia of the lower extremity. The STILE trial. Ann Surg 1994;220(3):251–266 DOI: 10.1097/00000658-199409000-00003
2. Illuminati G,Bresadola L,D'Urso A, et al. Simultaneous stent grafting of the descending thoracic aorta and aortofemoral bypass for "shaggy aorta" syndrome. Canadian Journal of surgery. Journal Canadien de Chirurgie 2007;50(5):E1–E2.
3. Wu MY,Yiang GT,Liao WT, et al. Current mechanistic concepts in ischemia and reperfusion injury. Cell Physiol Biochem 2018;46(4):1650–1667. DOI: 10.1159/000489241
4. Collard CD,Gelman S. Pathophysiology, clinical manifestations, and prevention of ischemia-reperfusion injury. Anesthesiology 2001;94:1133–1138. DOI: 10.1097/00000542-200106000-00030
5. Dorweiler B,Pruefer D,Andrasi TB, et al. Ischemia-reperfusion injury: pathophysiology and clinical implications. Eur J Trauma Emerg Surg 2007;33(6):600–612. DOI: 10.1007/s00068-007-7152-z